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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re application of: Alexander H. Borchers et al.

Serial No.: Not yet assigned Group No.: Not yet assigned

Filed: herewith

For: Antisense Modulation of ABC transporter MHC 1 Expression

BOX SEQUENCE

Assistant Commissioner for Patents
Washington DC 20231

INFORMATION DISCLOSURE STATEMENT

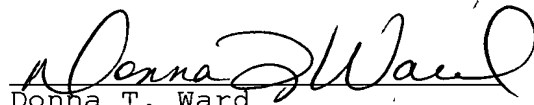
Pursuant to 37 C.F.R. §1.56 and in accordance with 37 C.F.R. §§1.97-1.98, information relating to the above-identified application is hereby disclosed. Inclusion of information in this statement is not to be construed as an admission that this information is material as that term is defined in 37 C.F.R. §1.56(b).

In accordance with §1.97(b), since this Information Disclosure Statement is being filed either within three months of the filing date of the above identified application, within three months of the date of entry into the national stage of the above identified application as set forth in §1.491, or before the mailing date of a first Office Action on the merits of the above identified application, no additional fee is required.

Copies of each of the references listed on the attached Form PTO-1449 are enclosed.

Date: December 17, 2001

Respectfully submitted,



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Form PTO-1449 Modified		Docket No. RTS-0353	Serial No. not yet assigned
List of Patents and Publications Cited by Application (Use several sheets if necessary)		Applicant Alexander H. Borchers et al.	
		Filing Date herewith	Group
U.S. Department of Commerce Patent and Trademark Office			
OTHER DOCUMENTS (Including Author, Title, Date, Pertinent Pages, Etc.)			
	AA	Alimonti et al., TAP expression provides a general method for improving the recognition of malignant cells in vivo, Nat. Biotechnol., 2000, 18:515-520	
	AB	Bahram et al., Two putative subunits of a peptide pump encoded in the human major histocompatibility complex class II region, Proc. Natl. Acad. Sci. U. S. A., 1991, 88:10094-10098	
	AC	Chen et al., A functionally defective allele of TAP1 results in loss of MHC class I antigen presentation in a human lung cancer, Nat. Genet., 1996, 13:210-213	
	AD	Colonna et al., Allelic variants of the human putative peptide transporter involved in antigen processing, Proc. Natl. Acad. Sci. U. S. A., 1992, 89:3932-3936	
	AE	de la Salle et al., HLA class I deficiencies due to mutations in subunit 1 of the peptide transporter TAP1, J. Clin. Invest., 1999, 103:R9-R13	
	AF	Furukawa et al., Splice acceptor site mutation of the transporter associated with antigen processing-1 gene in human bare lymphocyte syndrome, J. Clin. Invest., 1999, 103:755-758	
	AG	Galocha et al., The active site of ICP47, a herpes simplex virus-encoded inhibitor of the major histocompatibility complex (MHC)-encoded peptide transporter associated with antigen processing (TAP), maps to the NH2-terminal 35 residues, J. Exp. Med., 1997, 185:1565-1572	
	AH	Gorbulev et al., Allosteric crosstalk between peptide-binding, transport, and ATP hydrolysis of the ABC transporter TAP, Proc. Natl. Acad. Sci. U. S. A., 2001, 98:3732-3737	
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	AJ	Jackson et al., TAP1 alleles in insulin-dependent diabetes mellitus: a newly defined centromeric boundary of disease susceptibility, Proc. Natl. Acad. Sci. U. S. A., 1993, 90:11079-11083	
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	AK	Karttunen et al., Antigen presentation: TAP dances with ATP, Curr. Biol., 1999, 9:R820-824	
	AL	Klein et al., An inventory of the human ABC proteins, Biochim. Biophys. Acta, 1999, 1461:237-262	
	AM	Lage et al., Enhanced expression of human ABC-transporter tap is associated with cellular resistance to mitoxantrone, FEBS Lett., 2001, 503:179-184	
	AN	Li et al., Tapasin is required for efficient peptide binding to transporter associated with antigen processing, J. Biol. Chem., 2000, 275:1581-1586	
	AO	Ljunggren et al., MHC class I expression and CD8+ T cell development in TAP1/beta 2-microglobulin double mutant mice, Int. Immunol., 1995, 7:975-984	
	AP	Min et al., Interferon induction of TAP1: the phosphatase SHP-1 regulates crossover between the IFN-alpha/beta and the IFN-gamma signal-transduction pathways, Circ. Res., 1998, 83:815-823	
	AQ	Reits et al., The major substrates for TAP in vivo are derived from newly synthesized proteins, Nature, 2000, 404:774-778	
	AR	Sospedra et al., Hyperexpression of transporter in antigen processing-1 (TAP-1) in thyroid glands affected by autoimmunity: a contributing factor to the breach of tolerance to thyroid antigens?, Clin. Exp. Immunol., 1997, 109:98-106	
	AS	Spies et al., A gene in the human major histocompatibility complex class II region controlling the class I antigen presentation pathway, Nature, 1990, 348:744-747	
	AT	Takeuchi et al., Association of TAP1 and TAP2 with systemic sclerosis in Japanese, Clin. Exp. Rheumatol., 1996, 14:513-521	
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	AU	Takeuchi et al., Polymorphism of TAP1 and TAP2 in Japanese patients with rheumatoid arthritis, Tissue Antigens, 1997, 49:280-282	
	AV	van Endert et al., Genomic polymorphism, recombination, and linkage disequilibrium in human major histocompatibility complex-encoded antigen-processing genes, Proc. Natl. Acad. Sci. U. S. A., 1992, 89:11594-11597	
	AW	Van Kaer et al., TAP1 mutant mice are deficient in antigen presentation; surface class I molecules, and CD4-8+ T cells, Cell, 1992, 71:1205-1214	
	AX	Vives-Pi et al., Expression of transporter associated with antigen processing-1 in the endocrine cells of human pancreatic islets: effect of cytokines and evidence of hyperexpression in IDDM, Diabetes, 1996, 45:779-788	
	AY	Wong et al., Induction of primary, human antigen-specific cytotoxic T lymphocytes in vitro using dendritic cells pulsed with peptides, J. Immunother., 1998, 21:32-40	
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U.S. PATENT DOCUMENTS

Examiner's Initial		Document No.	Date	Name	Class	Subclass
	AA	6,087,122	7/11/2000	Hustad et al.	435	29
	AB	5,831,068	11/3/1998	Nair et al.	536	24.5
	AC					
	AD					
	AE					
	AF					
	AG					
	AH					
	AI					
	AJ					
	AK					
	AL					
	AM					
	AN					

FOREIGN PATENT DOCUMENTS

Examiner's Initial		Document No.	Date	Country	Translation YES NO	
	AO	WO 01/73027	10/4/2001	PCT	X	
	AP	WO 01/49716	7/12/2001	PCT	X	
	AQ					
	AR					
	AS					
	AT					
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	AV					
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	AX					

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